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# A Study of the Dog Electrocardiogram By the Method of Spatial Vector Analysis

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# A STUDY OF THE DOG ELECTROCARDIOGRAM BY THE METHOD OF SPATIAL VECTOR ANALYSIS

by

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## LIFE

James Graham Dobbie was born in Oak Park, Illinois, April 28, 1926.

He was graduated from Proviso Township High School, Maywood, Illinois, June 1944; entered the United States Navy July 1944; graduated from Northern Illinois University, June 1950 and from Marquette University School of Medicine, June 1955, and interned at Cook County Hospital 1955-1956.

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# CHAPTER I

## INTRODUCTION AND THE STATEMENT OF THE PROBLEM

The recording of the electrical activity of the heart from the surfaces of the body has been of great value in the study of the mammalian heart in health and disease. In experimental animals, a valuable pursuit is the production of definite cardiac lesions and the study of the electrocardiographic changes elicited thereby. Obviously, this could have important application in the study of cardiac abnormalities in man.

One of the most commonly used animals in cardiac research is the dog. At present, however, the interpretation of dog electrocardiograms (EKG) presents difficulties due to the day to day variability in the recorded electrocardiographic patterns. These variations are thought to be the result of the weak mediastinal support in the heart of the dog placed in the supine position which allows considerable movement of the heart with slight changes in position of the animal. Such movements produce rather marked variations in the EKG making comparison of serial EKGs quite difficult.

A procedure which allows interpretation of EKGs in terms of their spatial characteristics facilitates differentiation of abnormal variations from those due to positional changes alone. This method has been employed in the examination of the human heart but has never been used in experimental

animals. The purpose of this thesis was to investigate the use of spatial vector cardiography in the analysis of dog EKGs. Secondly, the normal dog EKG was compared with those taken from dogs having experimentally induced cardiac abnormalities.

## CHAPTER II

## REVIEW OF LITERATURE

### A. Effect of Body Position on the Human Electrocardiogram.

Advances in the field of electrocardiography have clarified and put on a rational basis observations on the effect of cardiac position on the various leads. Long ago investigators noted EKG changes in lateral recumbent positions, in extremes of body habitus and during respiration. Einthoven in 1900 noted that the P,Q,R waves were larger, and S and T waves smaller when the subject was placed in the right lateral than in the left lateral position, explaining that the heart rotated on its longitudinal axis in the chest when the patient turned. Later investigators showed that lateral displacement of the heart as in atelectasis or pleural effusion in itself did not affect the limb leads but rotation about the anterior-posterior (A-P) or long axis did (Dieuaide, 1925). Others have demonstrated left axis shifts with rotation of the heart to the left about the A-P axis as well as to the right about the long axis and right axis shifts with rotations in the opposite direction. Dieuaide (1927), in fact, proposed that the presence or absence of 'normal' shift of the electrical axis in lateral positions be used as a test for adhesive mediastino-pericarditis. However, Nathanson (1935) later showed that the right axis deviation occurred in 83 per cent of 'normal' subjects in the left lateral position, left axis deviations in 35 per cent of subjects in the right lateral position, and that

no change occurred in some 10 per cent of normal subjects.

The effect of body habitus was noted early. The transverse heart of obesity typically shows left axis deviation and the vertical heart of the tall thin subject, right axis deviation. These changes are quite constant, and the suggestion has been made by Cohn (1927) that when the EKG does not conform to the habitus of the patient, an anatomic change in the structure of the heart must be considered.

Rotations of the heart during respiration were studied by Samojloff in 1908 and many investigators since. With inspiration, the diaphragm descends and the heart becomes more elongated and vertical with a right axis shift noted in the limb leads; with expiration opposite changes are seen (Master, 1942). Analogous changes have been recorded after meals (Simonson, 1946, 1950), due to the distended stomach altering intrathoracic relations.

The reintroduction of unipolar and vector EKG has altered concepts of the electrical determination of cardiac position. Goldberger's monograph (1949) gives criteria for determination of cardiac position in the frontal plane from the unipolar limb leads. Wilson (1944) described five electrical positions of the heart based on the appearance of the V or AV leads in relation to the precordial leads. Correlation of the electrical position with the actual position of the heart as revealed by x-ray studies has been good in many (Hyman, 1948; Fowler, 1951: Rosenman, 1950; Kisten, 1950), but not all instances. It has been claimed that the position of the heart in the frontal plane as derived from the standard leads, unipolar limb leads, precordial and limb lead correlations may be determined fairly accurately, probably with an error of less than 10 per cent (Wilson, 1938).

With respect to the sagittal plane, there is much less agreement. Goldberger's criteria (1943) for forward and backward rotation have not been substantiated (Fowler, 1951). Loop vector cardiographic techniques (Wilson, 1938), however, give adequate representation of the sagittal plane, but the required equipment is complex. Grant has recently published (1950, 1951) a simple technique for estimation of electrical mean spatial vectors. The direction of the electrical vectors in the frontal plane are estimated from the limb leads, and their direction in the sagittal plane from the distribution of positive and negative complexes on the thoracic wall, as revealed by the precordial leads. Helm (1953) has recently developed a table which makes calculation of the spatial angle between the QRS and T mean vectors a simple matter.

B. Effect of Cardiac Lesions on the Human Electrocardiogram.

In an extensive review of the literature combined with his own experience, Katz (1947) stated that there are several characteristic patterns in the EKGs seen in congenital heart diseases. He describes the pathognomic changes seen in dextrocardia and also describes the patterns seen in conditions which place increased demand on the right or left heart ventricles. The pattern changes seen in these cases were defined as 'strain' patterns. He noted left ventricular strain patterns in aortic stenosis and right ventricular strain in pulmonary artery stenosis. A combined strain pattern was seen in patent ductus arteriosus.

C. Vectorcardiographic Studies in Congenital Heart Disease in Humans.

There have been relatively few studies of congenital heart disease in humans utilizing vectorcardiography. Freidberg (1956) states that attempts

to correlate hypertrophy patterns with congenital heart disease are not justified since the patterns are for the most part far from diagnostic. The findings of Kjellberg and Manheimer (1954) are in agreement with those of Freidberg.

Sapin (1956) discusses current use of vectorcardiography in the diagnosis of congenital cardiac malformation and concluded that its chief contribution is the determination of ventricular preponderance in mitral stenosis, pulmonary artery stenosis, and patent ductus arteriosus. This study was primarily with use of vector loops, however, it can be seen that the spatial angle is larger than normal in these cases of ventricular hypertrophy. Sapin's studies were in agreement with similar ones by Brawnwald (1956).

Elk (1954) studied vectorcardiograms in pulmonary stenosis and mitral stenosis and found that in these conditions the vectorcardiograms usually showed evidence of right ventricular preponderance as evidenced by the mean vectors pointing more anterior and to the right. There was no mention made of it but it could be seen that their tracings showed a wide spatial angle also.

Grant (1950) has reported on a 'normal range' of the spatial angle in humans and also observed increased spatial angles in myocardial infarction and ventricular hypertrophy. Greshman (1951) and Gubner's (1943) findings in ventricular hypertrophy are in agreement with those of Grant.

D. <u>Effect of Body Position on the Dog Electrocardiogram</u>.

The dog continues to be the most frequently used animal in experimental electrocardiography. As yet, there have been no reports in the

literature of the study of the dog EKG by the method of measuring spatial angles as used in this paper. The following survey deals, therefore, primarily with variations in electrocardiographic patterns.

Statements have been made in the literature to the effect that EKGs taken on dogs differ in some respects from human records and that EKGs which are considered pathologic in man may be observed fairly frequently under normal circumstances in the dog.

The observation that T waves may not infrequently be inverted in all three leads in normal dogs was first made by Smith (1918). The extreme variability in the T waves of normal dogs as contrasted to the T waves of normal human subjects was pointed out by Barnes and Mann (1931) who concluded that unless the direction of the T wave had been established under normal conditions in dogs, conclusions could not be drawn following cardiac insults.

Katz, Soskin, and Frish (1934) studied serial EKGs in normal, unanesthetized dogs over a period of four months and concluded that the variability in the T waves was caused by the relative mobility of the dog heart and suggested serial EKGs over a period of days to be carried out previous to the experimental EKG studies.

Harris and Hussey (1936) reported on the frequency and direction of T wave variations in 75 records on 50 normal dogs. Gross and Calef (1937) found that their normal records showed the T wave variations which had been reported by previous workers.

Peterson and Ricketts (1951) studied 32 normal dogs ranging in age from 5 to 28 months. These were untrained, unanesthetized animals in

which the EKGs were taken in the supine position. These workers found a mean QRS vector axis variation in repeated EKG of 4 degrees, and maximum variation of 8 degrees. They found that the T wave in lead I was usually iso-electric, and that T-II and T-III was always upright, and no change in the overall EKG pattern in any of the animals. They mentioned only whether or not the T waves were upright or inverted and whether they reversed their direction; nothing was said regarding the size of the deflection. Only 18 of the 32 animals had repeated tracings, and of these 18 only one had more than two tracings carried out. Lalich and Cohen (1941) found that T waves varied in amplitude and even reversed direction in the same animal on successive tracings taken daily over a period of several days. They found a QRS mean vector axis shift of as great as 75 degrees when the animals were changed in position from lying on their right to left side. The day to day variation averaged 15 degrees in the mean QRS axis which in over 60 per cent of the animals was located between 50 and 75 degrees. Horwitz and Wiggers (1953) found variations in the amplitude and duration of deflections in serial EKGs taken on normal dogs. They suggested the mean deflections of three different EKGs be taken as a control on each dog used for study.

On studies of animals following ligation of coronary arteries, Smith (1918) reported pattern changes similar to those found by us although his mortality was high and most of the cases reported were studied over a relatively short period of time. In the 12 of the 37 animals that lived long enough to obtain satisfactory tracings, several were found at autopsy to have hypertrophy of the right ventricle.

Harris (1936) in his EKG studies on dogs following coronary artery ligation found that the control group showed marked variation in the normal tracing patterns, and a more marked T wave variation and increased amplitude of T waves postoperatively.

Barnes (1931) found that the QRS pattern did not change much following ligation of the coronary arteries in dogs, and although the T waves changed considerably, they were thought by Barnes to be unreliable as an index of infarction because he felt that the T wave was notoriously unreliable even in the normal dog and comparison with the tracings taken following coronary artery ligation was extremely difficult.

Gross (1937) found that following coronary artery ligation in the dog, the ST segment deviated early and returned to the iso-electric line later. He felt that since there was no coronary artery sclerosis or myocardial fibrosis, the heart was able to compensate for the injury with the resultant return of the ST segment deviation to the iso-electric line.

In all of the preceding papers, the EKGs were done by the standard methods and standardization of the control EKGs was based on standardization of body position. This position may in some instances be impractical; furthermore, even when the dog was placed in apparently the same position, the relative position of the heart was not always the same during successive experiments. Even during the same experiment the electrographic pattern, as has been described by the workers mentioned previously, has been seen to change significantly while the dog remains in its original position. Because of this, other methods of standardizing the dog EKG is desirable. Recently the first attempt to apply loop vectorcardiography

to studies on dogs was carried out by Horon and Burch (1957). These authors describe some loops which were taken on normal dogs. This method is complex and not easily adapted to routine laboratory use. The mean spatial vectors as compared to the loop vector tracings were derived from the standard EKG. The mean vector, as the name implies, is a single summation product and does not show the numerous instantaneous forces which have combined to produce it. The loop vectorcardiogram is a more complete visualization of the electrical field of the heart derived with the cathode ray oscilloscope. By varying electrode positions, any plane may be inscribed, although by custom, the loops are usually determined in frontal, horizontal, and sagittal aspects. This technique would seem to give more information than the mean spatial vectors. However, at this time, the mean spatial technique is of greater value for the following reasons. Firstly, it utilizes standard electrocardiographic equipment, technique, and EKGs, no special equipment or technical assistance are needed. Secondly, there is as yet much disagreement among workers with the oscilloscopic technique as to the electrode placement and of other matters of method. Until a more uniform approach is agreed upon, this cannot be used effectively as a routine research tool. Along this line, the mean spatial technique is of special value because it serves as a connecting link between pattern interpretation and the oscilloscopic loops. Correct understanding of the principles of the mean spatial vector method enables one to correlate the two in a manner which is not otherwise possible; and should the time come when loop vectorcardiography becomes a more simple and routine matter, the findings obtained by use of the mean spatial vector method will fit nicely

into the whole picture.

E. Relationship of Spatial Angle and Ventricular Gradient.

The spatial angle is that angle bounded by the mean spatial QRS and T mean spatial vectors. The significance of the spatial angle has not yet been fully determined since it is a relatively recent measurement.

A discussion of the term ventricular gradient which Wilson introduced a number of years ago is relevant at this point since there is a relationship between the spatial angle and the ventricular gradient. The ventricular gradient is a more accurate measurement of the electrical forces of the heart since it measures both the magnitude and direction of the vectors. Numerous authors have pointed out, however, that T wave vectors in humans are altered primarily in their magnitude as a result of many systemic disorders which are of relatively little significance as far as the heart is concerned. Systemic diseases affect all of the myocardial cells relatively equally. Consequently, they produce a relatively symmetrical alteration in the T forces to the heart. This alters the magnitude of the mean spatial T vector significantly, but not its direction. Conversely, in those diseases which are characterized by significant cardiac damage, the heart is involved asymmetrically, one area more than the other. In this asymmetrical myocardial involvement, the T mean vector is altered in direction without change in its magnitude, consequently the spatial angle is altered most markedly in the more significant myocardial disease. The ventricular gradient method incorporates the magnitude as well as direction of the vectors in the calculation. This added factor deprives this method of some sensitivity in the diagnosis of organic heart disease, which the

calculation of the vector direction alone in the spatial angle measurement provides.

The nature of the spatial angle is related to the same phenomena observed in the measurement of the ventricular gradient. If the sequence of repolarization forces is the same as that of depolarization forces, there is no difference in magnitude and the direction is opposite. Subsequently there is no gradient between these vector forces. When the sequence of repolarization differs from that of depolarization, a gradient exists. This is called the ventricular gradient by Wilson.

There have been many attempts to explain the ventricular gradient. One theory is that there is a different sequence of repolarization and depolarization due to a delay in repolarization at the endocardial surface; this is due to increased mechanical tension in the walls of the ventricles brought on by contraction of the ventricles. Another explanation of the ventricular gradient may be due to the fact that there are different pathways taken by the depolarization and repolarization processes. The gradient might also be partly due to thermal changes within the cell during the greater metabolic activity associated with repolarization. The fact that there are several very different theories indicates that the true nature of the ventricular gradient has yet to be described. Since the measurement of the ventricular gradient is a more time-consuming and complex procedure, and less likely to reveal additional information, it was considered more significant to study the spatial angle in spite of a comparative lack of a clear understanding of the nature of either.

## CHAPTER III

## METHODS AND PROCEDURES

The animals used on our experiments were normal healthy dogs of similar size and weight. Of the 34 animals used, none were very young or extremely old. These are the same animals used by Flynn (1953) in studies on the hemodynamics of normal dogs and of those with surgically produced cardiac lesions.

In a typical experiment, EKGs were taken after the animal was tied to an animal board and had become quiet for several minutes. No anesthetic was used during this phase of the study and the animals soon learned to lie quietly while the EKGs were taken.

In the areas of the chest and limb leads, a small area of hair was shaved off close to the skin at the site of each lead. This allowed good contact of the electrode and also acted as a marker for subsequent electrode placement. It is important to place the electrodes in the same position in each experiment because, as will be seen later, variation of the electrode position can change the spatial angle. Good electrode contact was assured by use of Sanborn electrocardiographic electrode paste; however, any similar paste will suffice. The Sanborn poly-viso cardiette was employed for recording the EKGs. This is a four channel machine and from one to four tracings can be made simultaneously.

In experiments where fluid volume studies were carried out, the area over a leg vein was shaved and swabbed with alcohol after which the dye was injected and blood samples withdrawn. In studies in which cardiac output was determined, the animals were anesthetized with nembutal in the dosage of 30 mgms. per kilogram of body weight. The anesthetized animal was placed in the supine position on the animal board and was tied down. The area over the jugular vein was exposed, and a No. 8 French ureteral catheter was inserted through a small incision in the vein. After the procedures for the cardiac output measurement were completed, the catheter was removed and one or two 5-0 black silk sutures were used to close the defect in the vein and one 4-0 black silk suture closed the skin defect.

A. Production of Experimental Lesions.

The surgical procedures were carried out under nembutal anesthesia and with sterile technique. The anesthetized animals were tied to the animal board on their right side and the left chest wall was shaved and washed with Zephiran aqueous solution. An endotracheal tube was inserted and positive pressure artificial respiration was given throughout the entire procedure. A total of 250 to 500 cc. of 5 per cent dextrose solution was given intravenously during and immediately following surgery.

The left side of the thorax was opened through the fourth interspace and the fourth rib resected from its costochondral junction as far back toward the vertebral column as was accessible. With the animal on artificial respiration, incision of the pleura was made. The lung lobes were retracted and the heart within the pericardial sac, great vessels, vagus,

and left phrenic nerve were identified.

Five cc. of 5 per cent solution of procaine amide was injected into the pericardial sac. This solution was allowed to bathe the heart for at least three minutes before the pericardial sac was opened as a precautionary measure to prevent ventricular fibrillation.

The pericardial sac was incised and the flaps of pericardium held by sutures so as to form a sling for the heart and great vessels while the surgical procedures were being carried out on the pulmonary and coronary arteries. The mobilization of the pulmonary artery from the pericardium and fat pads is the most hazardous part of the operation; because of the extreme thinness of the pulmonary artery, great caution must be exercised in its manipulation particularly during the posterior and inferior dissection.

B. Pulmonary Stenosis.

One method used in an attempt to produce pulmonary stenosis for study in our series of abnormal dogs was by the use of polyethylene. This substance was first introduced by Poppe (1946) for surrounding an aneurism of the aorta with a reactive material capable of inducing a dense fibrous tissue response. Many investigators have since tried this procedure with varying success.

Hufnagel and Gillespie (1950, 1951) found that polyethylene in itself is not fibroblastic and postulated that the reactive material was a plasticizing material, dicetyl phosphate. Hufnagel postulates that fibrosis can be induced by finely divided piezo-electric crystals, i.e. crystalline materials of asymmetric structure. It is said that in each of such

crystals, minute electric currents are set up constantly by the physical stress placed upon them by the surrounding structures, and this stimulates the production of dense highly collagenous fibrous tissue. Using the barium salt of the dicetyl phosphate, Hufnagel has not been able to produce the dense fibrosis claimed by some investigators.

In an attempt to produce pulmonary stenosis, we have used both polyethylene film, DuPont N-V-7-14, containing dicetyl phosphate and the salt of dicetyl phosphate. The film was wrapped around the pulmonary artery and the dicetyl phosphate, which had been previously synthesized, was injected into the adventitia of the wall of the pulmonary artery. In no case was there any fibrotic response.

Further experiments were done by injecting the dicetyl phosphate in saline and corn oil suspensions into the peritoneal cavity of white rats. There was no more reaction to the dicetyl phosphate than there was to saline or corn oil at the end of six weeks. Attempts to produce pulmonary stenosis by this method were abandoned.

A second procedure for producing pulmonic stenosis was to completely isolate the artery and sew a wedge out of the artery just distal to the semilunar valves. This mechanical approach to a stenosis was very desirable in that it allowed the operator to stenose the artery until a dilatation of the right heart occurred at which time it was considered that sufficient constriction had been accomplished.

C. Avulsion of the Pulmonary Semilunar Valves.

Incomplete pulmonary semilunar valve avulsion was another method used to produce right heart failure and was carried out by the following

procedure. After complete dissection of the pulmonary artery intrapericardially, a large intestinal clamp was placed over the conus of the right ventricle just distal to the coronary artery and vein traversing the infundibular portion of the right ventricle. This was slowly closed so as to effectively cut off right heart output.

Since there was no blood being moved by the heart once the first clamp is closed, the following procedure was completed within two minutes. A second bowel clamp with rubber tips is placed 1½ to 2 cms. distal to the clamp around the right ventriculo-pulmonary artery junction. An incision is made into the dorsal surface of the pulmonary artery, isolated between the two clamps. The walls of the artery are retracted to give adequate exposure of the semilunar valves. With fine toothed forceps, the valves were picked up and resected from the wall of the pulmonary artery with scissors. Two of the valves are readily accessible, but the third valve posteriorly is difficult to dissect out and is usually left intact. After this procedure, a clamp is placed parallel to the incision so as to clasp both walls together. The bowel clamps are then removed from the conus and distal pulmonary artery and the heart is once again able to pump blood from the right heart. This procedure was done alone and in combination with a stenosis of the pulmonary artery distal to the valves in the area of incision. The incision and stenosis are then sewn with continuous suture.

D. <u>Arteriovenous Fistula</u>.

Arteriovenous fistula is another method for producing failure of the right heart. This was combined with the previous methods (either valvu-

lotomy of the semilunars alone or in combination with a stenosis). The arteriovenous fistula requires a complete and extensive dissection of the subclavian and left pulmonary arteries. The left pulmonary artery was effectively tied off during this procedure. Special coarctation forceps were used in holding the subclavian artery after it was resected and brought in close approximation to the left pulmonary artery. The open ends of the subclavian artery were sutured to the open ends of the left pulmonary artery. The suture material employed in this procedure was the atraumatic needle with 5-0 deknatal black silk. The anastomosis site was generally covered with a strip of muscle or gelfoam.

Some slight dilatation of the right ventricle was noted during the period when the left pulmonary artery was tied off, however, the dilatation disappeared as soon as the artery was untied. No pressure studies were carried out during the surgery.

E. Coronary Occlusion.

After the pericardial sac was opened in several experiments, the left coronary artery was ligated. The site of the ligation was immediately proximal to the last large branch from the anterior descending portion of the left coronary artery. A 3-0 black silk suture was passed under the artery and its accompanying vein with care not to include any of the ventricular musculature. Within a few minutes after tying the ligature, the anterior apical area of the heart was seen to take on a very obvious cyanotic appearance. It is at this time that hearts were seen to go into ventricular fibrillation in a large number of cases unless procaine was injected into the pericardial sac as previously described. After the use

of procaine was adopted, no cases of ventricular fibrillation resulted. Orias (1932) and Coelho and Rocheta (1929) noted that heart failure may occur following myocardial infarction from coronary ligation although ventricular fibrillation was most likely to occur. In all of the above procedures after the lesions have been produced the chest cavity was cleared of excess blood and the lungs inflated with increased positive pressure relieving areas of collapse. The third and fifth ribs were brought into approximation with sutures and the muscle layers of the chest wall are likewise sutured together. The skin was generally closed with interrupted sutures and the animal given 100,000 units of penicillin G intramuscularly daily for one week.

The pericardium was incompletely closed to allow for acute dilatation of the right ventricle. If the pericardial sac is tightly closed, the sudden dilatation of the right ventricle caused a picture similar to acute pericardial tamponade and the animals died within a period of minutes to a few hours postoperatively.

F. Electrocardiographic Leads.

The limb and chest leads used in this work are those commonly used in routine electrocardiography. The limb leads may be separated into the bipolar and unipolar leads. The bipolar limb leads were devised by Einthoven and in these leads he recognized a mathematical relationship which is now known as Einthoven's law. This law is simply an adaptation to electrocardiography of a well known principle of physics; when potentials are measured consecutively along a series of points, which return to the original point, the vector addition of all these potentials equals zero.

Einthoven chose to measure the potential in a particular sequence; first he measured the potential between the right arm and the left arm (lead I), then between left arm and left leg (lead III), and finally between left leg and right arm (lead II). Therefore, he began and ended his measurements at the right arm so that the QRS and T deflections of I plus II plus III equals 0. However, in composing the polarity of these leads, Einthoven reversed the polarity of lead II. Conceivably, this was done in order to have upright deflections in all three leads, a convention commonly observed by physicists. Because of this reversed polarity, the equation becomes lead I plus lead III plus (minus lead II) equals 0. This may be transposed into lead I plus lead III equals lead II which is the usual form of Einthoven's law.

The unipolar limb leads are recorded from exactly the same electrode positions as the bipolar limb leads. Therefore, the unipolar leads measure the electrical forces in the same plane in the body as do the bipolar leads. However, the galvanometer connections are different for the two types. The axes for the unipolar leads have directions which are different from those for the bipolar limb leads. In bipolar limb leads, the positive and the negative poles of the galvanometer are each connected directly to a limb electrode. In unipolar limb leads the negative pole of the galvanometer is connected to all three limb electrodes and the positive pole is connected to each of the three extremities in turn to record the lead for that extremity. Thus, in the unipolar circuit the negative electrode is placed at the zero point of the electrical field for each lead. The axis for such a lead is a line from the extremity to

the center of the heart in the frontal plane. The various unipolar leads may be described as follows: AVR, from the right arm to the negative electrode; AVL, from the left arm to the negative electrode, and AVF, from the left leg to the negative electrode. When the axes for all three unipolar limb leads are drawn, they form a triaxial system. When both unipolar and bipolar limb leads are taken, one has measurements of frontal plane vectors on six axes (Figure 1). This makes for greater accuracy in finding the mean vectors in the frontal plane.

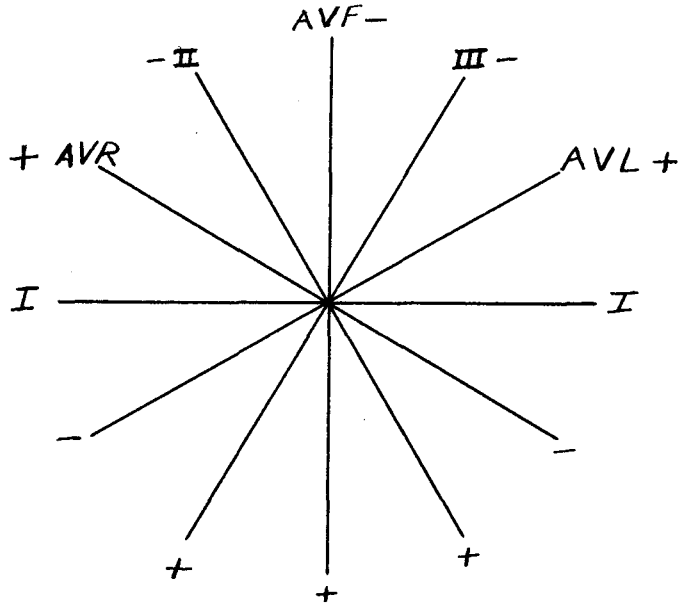


FIGURE 1

HEXAXIAL REFERENCE SYSTEM

In addition to the limb leads, other leads were taken from different positions on the anterior chest of the animals. These lead positions were in the form of a triangle and arranged in three rows. Chest lead I, C1, was taken just to the right of the midline approximately at the insertion of the third costal cartilage. C2 was taken at the same level and left

of the midline. The second row of chest leads is taken just inferiorly to the first row; C3 is placed slightly farther laterally than C1; C4 is in the same lateral position to the left of the midline, and C7 and C8 evenly spaced to the right and left of the midline. This arrangement gives approximately a triangular arrangement of the chest leads over the anterior aspect of the dog's chest (Figures 2A and 2B).

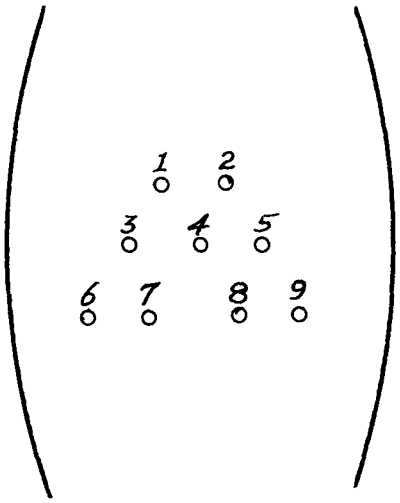


FIGURE 2A

CHEST ELECTRODE POSITIONS

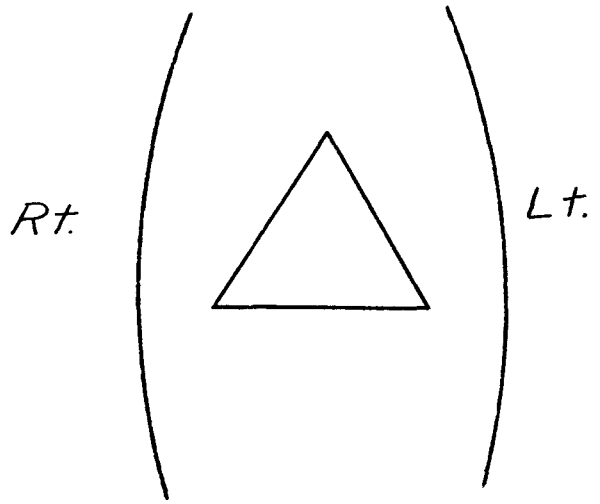


FIGURE 2B

FRONTAL PLANE OF THE DOG'S BODY

G. Planes of the Chest.

In order to avoid confusion, it is appropriate to describe the various planes of the chest. The frontal plane is that plane of the body parallel to the earth's horizon in an individual lying flat on his back. The sagittal plane parallels the earth's horizon when the individual is lying on his side and the transverse plane is parallel to the earth's horizon when the individual is in the upright position (Figure 3).

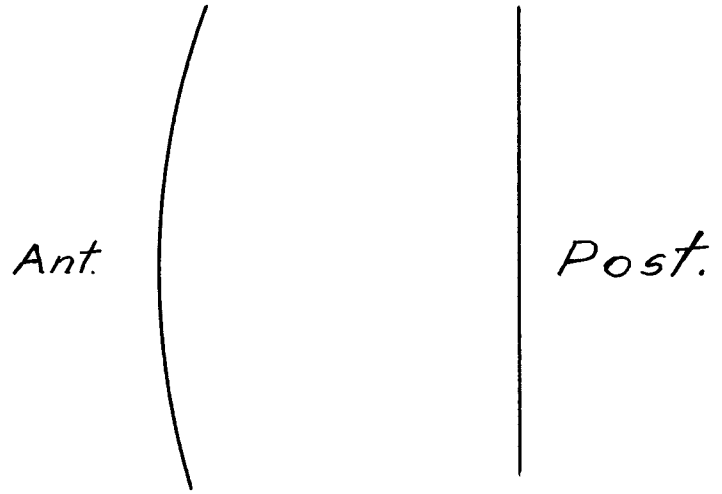


FIGURE 3

SAGITTAL PLANE OF THE DOG'S BODY

A diagram of the transverse plane may be made in the form of a T with the intersection of the lines forming the T representing the electrical center of the heart (Figure 4). The contour of the anterior chest wall is then drawn about the T as in Figure 5. The chest lead positions in which they are located on the dog are drawn in as in Figure 5. This diagram depicting the arrangement of the chest electrodes about the electrical center of the heart is used in the determination of the positions of vector projections on the transverse plane.

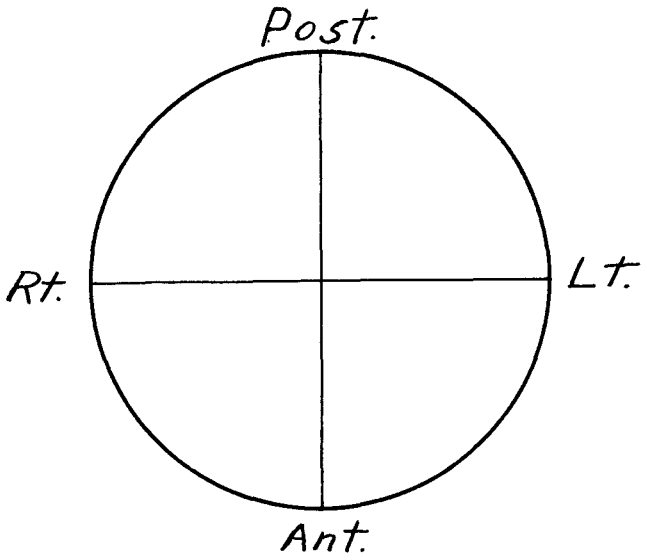


FIGURE 4

TRANSVERSE PLANE OF THE DOG'S BODY

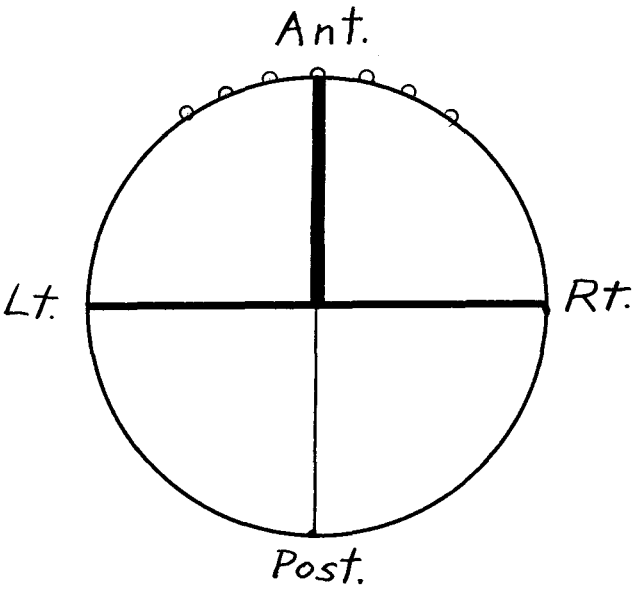


FIGURE 5

CHEST ELECTRODE DIAGRAM IN TRANSVERSE PLANE

H. Vectors.

The vector may be defined as a force having magnitude and direction. In this paper the vector force with which we are concerned is electrical; the magnitude and direction may be calculated. During the recording of the electrical activity from the heart, countless electrical vector forces are present at any instant.

The mean QRS and T vectors can be calculated for any of the previously mentioned three planes of the body, however, for the purposes of this paper, it is only necessary to determine these vectors in two planes, viz., the frontal and transverse planes.

In order to measure the vectors in the various planes, it is necessary to define certain reference points. In the frontal plane, the angular measurements are made in a clockwise direction with the zero reference at the subject's left hand (Figure 6).

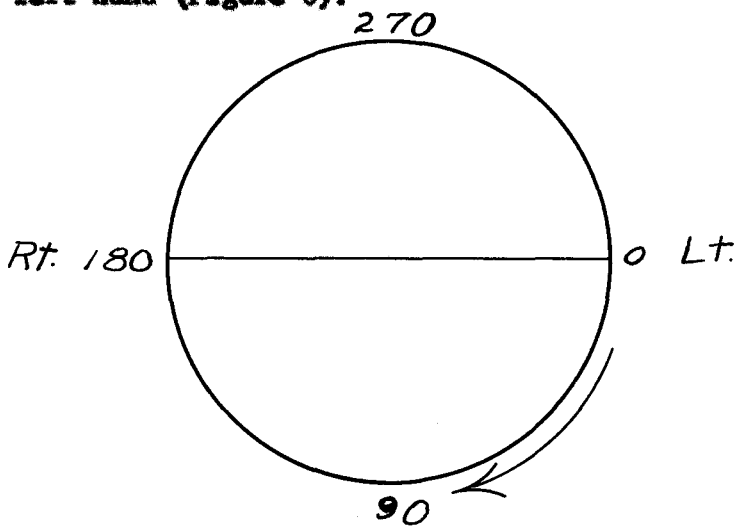


FIGURE 6

FRONTAL DIAGRAM OF VECTOR ANGLE IN DEGREES

In the transverse plane viewed from a point corresponding to the top of the head, angular measurements are also made in a clockwise direction from the posterior end of the anteroposterior axis as in Figure 7.

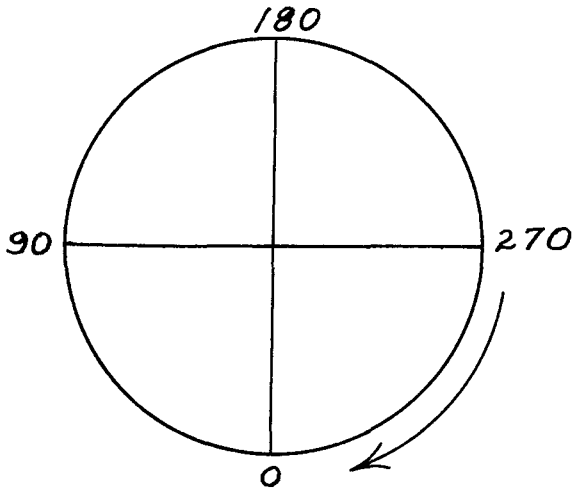


FIGURE 7

TRANSVERSE PLANE DIAGRAM OF VECTOR ANGLE IN DEGREES

The measurements of the vectors in both the frontal and transverse planes may be made through a range of 360 degrees.

I. Determination of Mean Spatial Vectors and Spatial Angles.

The aim of these calculations is to determine the mean electrical vectors of the heart in each of three planes corresponding to the geometric axes and from these values to determine the spatial angle. The latter is defined as the angle formed by the means QRS and mean T spatial vectors.

The most accurate way in which to calculate the mean frontal plane vectors is to measure the enclosed area under each portion of the deflection on each lead. This method would be too time-consuming and exacting for

routine use. However, the magnitudes of the vectors are not nearly as important as their directions, and the direction of a frontal plane vector can be determined with considerable accuracy from simple inspection of the limb leads. This simpler method depends upon the fact that when a vector is perpendicular to the axis of a particular lead, the deflection will be minimal in amplitude. In this way, the direction of the mean QRS and T vectors in the frontal plane can be determined from simple inspection of the limb leads, generally with less than 5 degrees of error.

In a similar manner the mean vectors may be determined in the transverse plane. Since the heart has a slightly greater mass to the left of the mediastinum, the electrical center of the heart is found slightly to the left of the midline (Schaften, 1953). The diagram used in the determination of the transverse plane vectors is seen as Figures 5 and 7.

Once the mean vectors have been drawn in both the transverse and frontal planes, it is a simple matter to determine the spatial angle from tables which are now available. Without getting into the complexities of the solid geometry involved, it may suffice to say that the procedure used allows calculation of the spatial angle from the determination of the mean QRS and T vector axes in both the frontal and transverse planes. The method for the use of these tables will now be described. For the QRS vector, the angle measured in the transverse plane is located in the horizontal marginal column of the table devised by Helm (1953), and the angle measured in the frontal plane is located in the vertical marginal column at the left of this table. The three numbers located at the intersection of the columns are read directly from the table. These three numbers represent the cosines of the angles

made by the mean vectors with the three geometrical axes. Positive or negative signs are given to these three values according to the signs listed in parentheses in the marginal columns. The two signs obtained from the horizontal marginal column are placed before the upper and middle numbers respectively. The two signs obtained from the vertical marginal column are placed before the middle and lower numbers respectively. Thus, the sign of the middle number may be obtained in two different ways. Failure of these signs to coincide indicates some defect in the angular measurements.

The angles measured in the transverse and frontal planes for the T vector are likewise determined and located in the horizontal and vertical marginal columns, respectively. In the same manner, the three values located at the intersection of these columns are read from Helm's Table I and the proper signs given to each.

Two sets of three values are thus obtained, one set for each vector. The respective products of the paired upper values, of the paired middle values, and of the paired lower values are obtained by multiplication. Proper signs are given to these three products in accordance with the rule that multiplication of numbers with like signs yields a product which is positive and multiplication of numbers with unlike signs yields a product which is negative. The three products are then added algebraically according to their signs. The result is the cosine of the angle between the two spatial vectors. In Helm's Table I, cosines are listed and the spatial angle may be read to the nearest 5 degrees. If the calculated cosine has a negative value, the angle chosen must be between 90 and 180 degrees.

In order to satisfy ourselves as to the reliability of Helms's method, we constructed a transparent model of the chest. Upon this the electrode positions were located in the frontal, transverse, and sagittal planes. Wooden arrows were placed within the hollow model, and the directions of the QRS and T vectors were laboriously reconstructed. It was found that Helm's tables gave very satisfactory and accurate results with relative simplicity and speed once the method was mastered. A sample calculation from Helm's table is shown below:

TABLE I

SAMPLE CALCULATION FROM HELM'S TABLE *

| | Transverse | Frontal |
|---|---|---|
| QRS | 55° | 330° |
| T | 135° | 15° |

(1) $\neq .52 \times -.69 = -.3588$
(2) $\neq .74 \times \neq .69 = \neq .5106$
(3) $-.43 \times \neq .19 = -.0817$

$\neq .0701$ = cosine of spatial angle

Spatial angle = 85°

* The reader is referred to the paper of R. A. Helm (1953) for a more complete description of the use of the above table.

## CHAPTER IV

## RESULTS

A. Range of the Spatial Angles.

The mean QRS and T vectors in the frontal and horizontal planes and the spatial angles on 35 normal dogs in the supine positions are shown in Table II. In the frontal plane, the average QRS vector is directed 71 degrees downward from the reference zero which is at the left arm, as described earlier. This direction is familiar in that it is the same as that plotted by freshman medical students from the standard limb leads. It is interesting to note that the angle of the average T vector in the same frontal plane is pointed toward the perpendicular although the vector is now pointing slightly toward the head in the head to tail axis. As is generally emphasized in basic electrocardiography, the electrical vectors do not necessarily bear relationship to the anatomical direction of the heart. In other words, the mean electrical vector is a composite of many considerations such as movements along linear and rotational axes, pathways of current travel in the tissue and variations in electrical activity in different areas of the organ.

In the horizontal plane, the average QRS vector is directed 161 degrees from the reference zero which is at the back of the body. Thus, the vector happens also to be 71 degrees from the left arm position although we are now speaking of the horizontal plane. The T vector appears shifted toward the anteroposterior axis so that the T vector is pointing directly towards the

back of the animal.

The spatial vector, which is a concept avoided in the previous discussion, is simple to describe here for the T variation since it is the resultant of a vector pointing towards the head in the frontal plane and a vector pointing towards the back in the horizontal plane; i.e., the spatial T vector points backward and upward in the sagittal plane. The exact angle in the sagittal plane is undetermined since the magnitude of the vectors, i.e. the length of the arrows, has not been evaluated. This determination is, however, unnecessary for the calculation of the spatial angle which is defined as the angle made by the spatial QRS vector and the spatial T vector. Under the conditions of our measurement, the spatial angle is determined as the angle made by the two planes in which the spatial vectors are located. The average spatial angle in 35 experiments has been found to be 26 degrees. It may be noted that the range of values for each of the measurements in Table II is extensive. In all out of the 35 cases, the values appear to be within twice the standard deviation of the mean spatial angle.

D. The Effect of Body Position.

In 6 experiments, animals were placed successively on their right side, in the supine position, and finally on their left side. Examination of Table III shows that there was no marked difference in the measurements made in the different body positions. This is somewhat surprising since it is generally believed that wide fluctuations occur in the dog due to displacement of the heart as body position is shifted. It should be pointed out, however, that there was considerable variation in the values measured in the supine position in the larger population shown in Table II.

TABLE II

MEAN FRONTAL AND HORIZONTAL QRS AND T VECTORS
AND SPATIAL ANGLES IN NORMAL DOGS

| Dog No. | Frontal QRS | Frontal T | Horizontal QRS | Horizontal T | Spatial Angle |
|---|---|---|---|---|---|
| 1 | 75 | 85 | 170 | 130 | 20 |
| 2 | 60 | 85 | 125 | 175 | 40 |
| 3 | 55 | 75 | 120 | 165 | 35 |
| 4 | 95 | 80 | 190 | 170 | 25 |
| 5 | 85 | 85 | 130 | 175 | 40 |
| 6 | 70 | 95 | 135 | 185 | 35 |
| 7 | 75 | 90 | 130 | 165 | 20 |
| 8 | 55 | 95 | 165 | 185 | 30 |
| 10 | 110 | 100 | 195 | 185 | 15 |
| 11 | 75 | 100 | 145 | 190 | 40 |
| 12 | 60 | 105 | 155 | 200 | 35 |
| 13 | 65 | 95 | 160 | 185 | 20 |
| 14 | 110 | 105 | 205 | 190 | 20 |
| 16 | 60 | 55 | 170 | 180 | 20 |
| 17 | 55 | 95 | 160 | 185 | 30 |
| 22 | 65 | 95 | 160 | 190 | 35 |
| 23 | 65 | 75 | 165 | 180 | 35 |
| 24 | 55 | 70 | 160 | 170 | 15 |
| 25 | 95 | 80 | 190 | 170 | 25 |
| 26 | 75 | 95 | 160 | 185 | 20 |
| 29 | 70 | 80 | 155 | 170 | 15 |
| 32 | 65 | 75 | 165 | 180 | 35 |
| 36 | 70 | 95 | 170 | 185 | 25 |
| 37 | 55 | 75 | 150 | 170 | 20 |
| 51 | 65 | 50 | 155 | 165 | 30 |
| 52 | 50 | 85 | 150 | 175 | 25 |
| 53 | 70 | 85 | 160 | 175 | 15 |
| 54 | 55 | 75 | 170 | 180 | 20 |
| 55 | 70 | 95 | 160 | 190 | 30 |
| 56 | 65 | 95 | 165 | 190 | 40 |

(continued)

TABLE II

(continued)

MEAN FRONTAL AND HORIZONTAL QRS AND T VECTORS
AND SPATIAL ANGLES IN NORMAL DOGS

| Dog No. | Frontal QRS | Frontal T | Horizontal QRS | Horizontal T | Spatial Angle |
|---|---|---|---|---|---|
| C1 | 105 | 125 | 195 | 205 | 20 |
| C2 | 120 | 110 | 200 | 190 | 15 |
| C3 | 70 | 95 | 160 | 195 | 35 |
| C4 | 65 | 75 | 155 | 165 | 15 |
| C5 | 60 | 85 | 165 | 175 | 25 |
| Mean | 71 | 87 | 161 | 179 | 26 |
| Range | (50-120) | (50-125) | (120-205) | (130-205) | (15-40) |

## TABLE III

## VECTOR SHIFTS WITH CHANGES OF BODY POSITION

| | Frontal Plane | | Horizontal Plane | | Spatial Angle |
|---|---|---|---|---|---|
| Dog No. | Mean QRS Vector | Mean T Vector | Mean QRS Vector | Mean T Vector | |
| | | | Right Side | | |
| 1 | 80 | 90 | 150 | 135 | 20 |
| 2 | 60 | 90 | 130 | 185 | 40 |
| 5 | 65 | 100 | 145 | 185 | 40 |
| 6 | 70 | 100 | 130 | 195 | 35 |
| 7 | 70 | 80 | 130 | 155 | 15 |
| 8 | 70 | 100 | 155 | 190 | 25 |
| Mean | 69 | 93 | 140 | 174 | 29 |
| Range | 60-80 | 80-100 | 130-155 | 135-195 | 15-40 |
| | | | Supine | | |
| 1 | 75 | 85 | 170 | 130 | 20 |
| 2 | 60 | 85 | 125 | 175 | 40 |
| 5 | 85 | 85 | 130 | 175 | 40 |
| 6 | 70 | 95 | 135 | 185 | 35 |
| 7 | 75 | 90 | 130 | 165 | 20 |
| 8 | 55 | 95 | 165 | 185 | 30 |
| Mean | 70 | 89 | 142 | 169 | 30 |
| Range | 55-85 | 85-95 | 125-170 | 130-185 | 20-40 |
| | | | Left Side | | |
| 1 | 75 | 70 | 155 | 125 | 20 |
| 2 | 65 | 90 | 130 | 185 | 35 |
| 5 | 80 | 85 | 130 | 175 | 40 |
| 6 | 70 | 105 | 145 | 195 | 40 |
| 7 | 70 | 80 | 130 | 150 | 15 |
| 8 | 50 | 85 | 160 | 175 | 30 |
| Mean | 68 | 86 | 142 | 168 | 30 |
| Range | 50-80 | 70-105 | 130-160 | 125-195 | 15-40 |

It may be noted that the shift in spatial angle is generally less than the shift in any of the mean vectors, i.e., the shift in the frontal and horizontal T vectors was 7 and 6 degrees, respectively, whereas the average variation in spatial angle was less than the shift in vector angle as the animal was shifted from the right to the left side. This indicates that the spatial angle is less affected by change in body position than the vector angle.

C. The Effect of Experimental Cardiac Lesions.

Spatial angles were determined in nine dogs before and after pulmonary stenosis. In Table IV, it may be noted that all animals had postoperative spatial angles greater than their preoperative values. In most cases, the change in the spatial angles appeared to be relatable to the T vector since it changed more consistently in one direction whereas the Q vector did not. This seemed more pronounced in the frontal plane.

In four animals that had stenosis of the pulmonary artery and avulsion of the pulmonary semilunar valves, the spatial angles increased except in one dog. In this case, the T vector in both planes showed a decrease which is highly unusual since in the group with stenosis only the T vectors either increased or remained the same. Although the QRS vectors appeared to decrease in all but one case, the variability seen in the stenosis group would appear to suggest that a larger number of experiments is necessary before this result is considered seriously.

TABLE IV

COMPARISON OF PREOPERATIVE AND POSTOPERATIVE VECTORS AND SPATIAL ANGLES

| Dog No. | Frontal Plane | | | | Horizontal Plane | | | | Spatial Angle | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Before | | After | | Before | | After | | Before | After |
| | QRS | T | QRS | T | QRS | T | QRS | T | | |
| **Stenosis** | | | | | | | | | | |
| 4 | 95 | 80 | 55 | 255 | 190 | 170 | 130 | 205 | 25 | 100 |
| 8 | 55 | 95 | 85 | 230 | 165 | 185 | 175 | 195 | 30 | 70 |
| 11 | 75 | 100 | 85 | 125 | 145 | 190 | 160 | 190 | 40 | 65 |
| 14 | 110 | 105 | 65 | 225 | 205 | 190 | 140 | 195 | 20 | 85 |
| 17 | 55 | 95 | 65 | 285 | 160 | 185 | 155 | 175 | 30 | 65 |
| 36 | 70 | 95 | 50 | 190 | 170 | 185 | 155 | 205 | 25 | 60 |
| 51 | 65 | 50 | 65 | 230 | 155 | 165 | 170 | 195 | 30 | 50 |
| 52 | 50 | 85 | 70 | 95 | 150 | 175 | 165 | 190 | 25 | 40 |
| 53 | 70 | 85 | 45 | 310 | 160 | 175 | 150 | 175 | 15 | 45 |
| Mean | 71 | 87 | 65 | 216 | 166 | 180 | 155 | 191 | 26 | 64 |
| Range | 50-110 | 50-105 | 45-85 | 95-310 | 145-205 | 165-190 | 130-175 | 190-205 | 15-40 | 40-100 |
| **Stenosis and Avulsion** | | | | | | | | | | |
| 4 | 95 | 80 | 70 | 290 | 190 | 170 | 160 | 175 | 25 | 60 |
| 8 | 55 | 95 | 70 | 235 | 165 | 185 | 130 | 200 | 30 | 105 |
| 17 | 55 | 95 | 50 | 40 | 160 | 185 | 150 | 160 | 30 | 20 |
| 36 | 70 | 95 | 60 | 230 | 170 | 185 | 135 | 200 | 25 | 75 |
| Mean | 69 | 93 | 62 | 199 | 171 | 181 | 144 | 183 | 27 | 65 |
| Range | 55-95 | 80-95 | 50-70 | 40-290 | 160-190 | 170-185 | 135-160 | 160-200 | 25-30 | 20-105 |
| **Fistula Alone** | | | | | | | | | | |
| 6 | 70 | 95 | 65 | 95 | 135 | 185 | 155 | 190 | 35 | 30 |
| 22 | 65 | 95 | 60 | 95 | 160 | 190 | 155 | 190 | 35 | 40 |
| 26 | 75 | 95 | 65 | 115 | 160 | 185 | 150 | 195 | 20 | 40 |
| Mean | 70 | 95 | 63 | 102 | 152 | 187 | 153 | 192 | 30 | 37 |
| Range | 65-75 | 95 | 60-65 | 95-115 | 135-160 | 185-190 | 150-155 | 190-195 | 20-35 | 30-40 |

(continued)

TABLE IV

(continued)

COMPARISON OF PREOPERATIVE AND POSTOPERATIVE VECTORS AND SPATIAL ANGLES

| Dog No. | Frontal Plane | | | | Horizontal Plane | | | | Spatial Angle | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Before | | After | | Before | | After | | Before | After |
| | QRS | T | QRS | T | QRS | T | QRS | T | | |
| **Stenosis and Fistula** | | | | | | | | | | |
| 11 | 75 | 100 | 60 | 75 | 145 | 190 | 155 | 180 | 40 | 45 |
| 23 | 65 | 75 | 70 | 95 | 165 | 180 | 165 | 190 | 35 | 35 |
| Mean | 70 | 87 | 65 | 85 | 155 | 185 | 160 | 185 | 37 | 40 |
| Range | 65-75 | 75-100 | 60-70 | 75-95 | 145-165 | 180-190 | 155-165 | 180-190 | 35-40 | 35-45 |
| **Stenosis, Avulsion and Fistula** | | | | | | | | | | |
| 14 | 110 | 105 | 65 | 115 | 205 | 190 | 150 | 195 | 20 | 40 |
| 51 | 65 | 50 | 75 | 115 | 155 | 165 | 165 | 200 | 30 | 30 |
| 52 | 50 | 85 | 65 | 110 | 150 | 175 | 170 | 200 | 25 | 40 |
| 53 | 70 | 85 | 55 | 340 | 160 | 175 | 150 | 160 | 15 | 45 |
| 54 | 55 | 75 | 65 | 260 | 170 | 180 | 160 | 190 | 20 | 85 |
| 55 | 70 | 95 | 55 | 250 | 160 | 190 | 155 | 190 | 30 | 70 |
| 56 | 65 | 95 | 110 | 170 | 165 | 190 | 200 | 290 | 40 | 85 |
| Mean | 69 | 84 | 70 | 194 | 166 | 180 | 164 | 203 | 25 | 56 |
| Range | 50-110 | 50-105 | 55-110 | 110-340 | 150-205 | 165-190 | 150-200 | 160-290 | 15-40 | 30-85 |
| **Coronary Occlusions** | | | | | | | | | | |
| 1 | 105 | 125 | 95 | 165 | 195 | 205 | 195 | 205 | 20 | 70 |
| 2 | 120 | 110 | 150 | 330 | 200 | 190 | 220 | 30 | 15 | 165 |
| 3 | 70 | 95 | 70 | 110 | 160 | 195 | 20 | 190 | 35 | 110 |
| 4 | 65 | 75 | 95 | 245 | 155 | 165 | 145 | 200 | 15 | 110 |
| 5 | 60 | 85 | 55 | 260 | 165 | 175 | 350 | 195 | 25 | 130 |
| Mean | 84 | 98 | 93 | 222 | 175 | 186 | 186 | 124 | 22 | 117 |
| Range | 60-120 | 75-125 | 55-150 | 110-330 | 155-200 | 165-205 | 20-350 | 30-205 | 15-35 | 70-165 |

Three animals were studied in which the cardiovascular lesion consisted of an arteriovenous fistula between the subclavian artery and the left pulmonary artery. The purpose of this lesion was to increase the work of the left heart. In only one of these animals was there significant increase in the spatial angle. In these animals it was felt that the heart may have compensated for the lesion.

Two animals were studied in which an arteriovenous fistula was combined with a pulmonary stenosis. The postoperative spatial angle was not found to be significantly greater than the preoperative spatial angle. The intracardiac pressure studies showed no appreciable difference in pressures when compared with the control pressure readings in these same animals (Flynn, 1953). This would indicate that the heart had compensated for the lesions.

A total of seven animals was studied in which the cardiovascular lesions induced were a pulmonary stenosis, pulmonary semilunar valve avulsion and an arteriovenous fistula. The surgical procedures were carried out in three steps: the pulmonary stenosis, the valvular avulsion, and lastly, the fistula, with sufficient time for recovery between operations. Increase in spatial angle was observed in six out of seven animals. The T vector showed a generally consistent increase following the lesions. Spatial angles were determined over a period of time and a decrease in magnitude was seen with the passage of time. This was attributed to the compensation by the heart to the surgical insults and, presumably, to a decrease in activity of these animals which lowered the demand on the heart. No exercises were carried out with the animals to determine their exercise tolerance so that it can only be presumed that these hearts were under physiologic stress.

The final group of animals studied was that in which the apical portion of the anterior descending coronary artery was tied off. In five animals, the spatial angles all increased. The T vectors also appeared to increase in this series.

## CHAPTER V

## DISCUSSION

### A. Principal Results of this Work.

Heretofore, interpretation of the dog EKG has been based primarily on the analysis of patterns in the EKG tracings. One of the most disturbing factors in dog electrocardiographic studies has been the fact that these 'patterns' change from day to day and even over a period of minutes in some cases. There has been no way in which to obtain a dog EKG that could be used as a control and would be reproducible from day to day. This paper describes a method whereby a reproducible measurement can be made which is independent of most of the factors which have made dog EKG interpretation difficult. That is, it provides a constant for the laboratory worker with each animal acting as its own control. This has not been accomplished previously in the dog. In addition, a normal range of spatial angles in a fairly large group of dogs was established.

The spatial angle measurement offers distinct advantages over the method of 'pattern' interpretation. Firstly, it is relatively simple, the mean QRS and mean T spatial vectors being the only two variables upon which interpretation is based. Secondly, it enhances the accuracy of interpretation of the various leads since it incorporates the deflection characteristics of all the limb and precordial leads into the evaluation. Thirdly, the spatial angle is a semi-quantitative measurement of the electrical forces

of the heart. Therefore, the study of follow up tracings and comparative analyses of various other tracings now becomes more objective than by use of other methods.

An important control study which needs to be investigated is the effect of different body positions on the spatial angle. In the human, no effect of body position has been noted (Grant, 1951). However, in dogs, where the loose mediastinal support allows the heart to move significantly within the chest cavity, the effect of body position on the spatial angle should be studied. This was done as part of our study and it was found that the effects of variations in body position upon the spatial angle are practically eliminated from interpretation provided the myocardium remains unchanged. This is not surprising since spatial angle measurements are independent of body position as well as the position of the heart within the chest cavity. This is explained by the fact that factors responsible for the spatial angle are intrinsic to the heart itself. We have observed marked changes in patterns on serial EKGs just as others have; this might be expected since the heart does shift within the chest cavity. However, the spatial angle remained significantly constant despite the pattern variations.

In our studies of normal animals, we did not see as great a variation in the frontal mean vectors as we anticipated. On careful analysis of the vectors, they were not found to vary as greatly with position variations as those studied by Conway (1949). However, our studies were done on a very much larger series of animals in which a few did show marked variation of the frontal vectors. It is felt that the reason Conway (1949) found such a significant variation in the vectors was due to the fact that they based

their conclusions on such a very few experiments.

As the reader has very likely observed, it is the T vector which shifts most markedly while the QRS vector remains relatively more stable. This is explained in the following paragraphs.

The QRS and T deflections represent two relatively different electrical processes in the heart. The electrical force which is responsible for the T wave is manifested when the myocardial cell metabolism causes the membranes to become relatively impermeable to cations, and the cations and anions are forced to line themselves on opposite sides of the membrane. The movement of the ions into this position on the cell membranes writes the T wave. When the cell loses its relative cation impermeability, the QRS complex is formed. It may then be said that the T wave is due to the repolarization, and the QRS complex due to the depolarization of the cell membrane.

Because the T force is produced by metabolic activity of the myocardial cell, the T deflection may be altered by physiologic and pathologic processes which have no demonstrable histologic effect on the heart muscle. The QRS force represents the 'discharge' of the polarized cells when an extrinsic impulse arrives at the membrane. Cell metabolism plays little part in the movement of the charges at this time and, therefore, factors which alter the T deflection may have no effect on the QRS deflection. This greater stability of the QRS force may be used as a kind of standard against which to compare the characteristics of the T force and in this way quantitate the changes which have taken place in the T force. This was seen repeatedly in our series of 'abnormal' animals, and the above is the explanation for this observation. From this, it may also be seen that if the myo-

cardium remains unchanged, the spatial angle will be the same no matter what degree of shift or rotation the heart may have undergone.

B. Usefulness of the Spatial Angle Measurements.

The spatial angle method of EKG interpretation is of greatest value in its simplicity. It greatly simplifies clinical interpretation of the EKG by eliminating the need for memorizing patterns. With the great number of different leads that are being taken in clinical and experimental electrocardiography, the memorizing of the abnormal and normal patterns for each lead has become nearly impossible for all but the specialist in electrocardiography. With the spatial angle method, the criteria for the normal and abnormal tracings become more simple and precise, which makes the evaluation of questionably normal and serial tracings easier and more accurate. Since the spatial angle method is based on a few relatively simple quantitative and objective measurements of the electrical activity in the heart, this method immediately becomes more accurate and objective than pattern interpretation. Furthermore, as previously indicated, changes in wave forms due to change in the position of the heart are easily separated from changes due to intrinsic myocardial abnormalities by use of the spatial angle method. There is absolutely no satisfactory way to make this differentiation with certainty in pattern methods of interpretation. Finally, the tracings taken from entirely unfamiliar regions of the body such as esophageal or intracardiac leads, may be interpreted accurately by the average non-specialized electrocardiographer.

C. Effect of Cardiac Lesions on the Vectorcardiogram.

In our studies on the effects of cardiac lesions on the vectors of the

heart, we first established a normal range of the spatial angles. Our normal range agreed very closely with those of Grant (1951), who has written and carried out a great amount of research on this phase of the subject carried out on human subjects. In subsequent studies, Grant (1951), reported on the changes seen in the vectorcardiogram and spatial angles of human cases of myocardial infarction. In his studies, the spatial angle increased greatly. In our experimental lesions in which the coronary arteries were tied off, we also noted changes similar to those observed by Grant.

Greshman (1951) reported vectorcardiographic studies in ventricular hypertrophy due to various causes, as did Sapin (1956), Lasser (1951), and Elek (1954). They found that regardless of the etiology of the hypertrophy there were alterations in the vectors which could be seen in almost all the cases studied. These findings were in agreement with the findings reported in this paper.

Kats (1947) reported on the patterns in congenital pulmonary stenosis and patent ductus arteriosus, conditions similar to our surgically induced cardiac lesions. Our patterns did, in some instances, show changes similar to his; however, we were primarily concerned with the vector interpretations and did not base our judgment of abnormal EKGs on the patterns.

It may be said that our findings are comparable with those most commonly found by other workers who have made similar studies in human electrocardiography.

## CHAPTER VI

## SUMMARY AND CONCLUSIONS

1. EKGs of normal dogs were examined by the method of spatial vector analysis, which is a procedure not previously employed in this animal. A normal range for the spatial angle in the dog EKG was established.

2. It was shown that the spatial angle of the T and QRS vectors is unchanged by body positional changes in the dog.

3. The following surgical insults to the heart and great vessels were carried out: a) pulmonary stenosis, b) pulmonary stenosis combined with pulmonary semilunar valve avulsion, c) arteriovenous fistula between the subclavian artery and the left pulmonary artery, d) pulmonary stenosis combined with arteriovenous fistula as in (c), e) pulmonary artery stenosis combined with pulmonary semilunar avulsion, and arteriovenous fistula, and f) coronary artery occlusion.

4. Dogs with pulmonic stenosis showed a significant increase in the spatial angle as compared to the preoperative spatial angles. The majority of the animals with pulmonary stenosis and pulmonary semilunar valve avulsion had spatial angles significantly greater than preoperative values.

5. Of the animals with an arteriovenous fistula, only one showed an angle significantly greater than its preoperative angle and even this was within the normal range. Of the animals in the stenosis, avulsion-fistula group, the majority showed a significant increase in the spatial angle. The

angles in this group were among the widest of any of the groups studied.

6. All of the animals in the group with coronary arterial occlusion showed a significant increase in the spatial angle. The angles in this group were by far the widest of any of the groups studied.

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## APPROVAL SHEET

The thesis submitted by James Graham Dobbie has been read and approved by three members of the Department of Physiology.

The final copies have been examined by the director of the thesis and the signature which appears below verifies the fact that any necessary changes have been incorporated, and that the thesis is now given final approval with reference to content, form, and mechanical accuracy.

The thesis is therefore accepted in partial fulfillment of the requirements for the Degree of Master of Science.

May 23, 1958
Date

Walter C. Randall
Signature of Advisor